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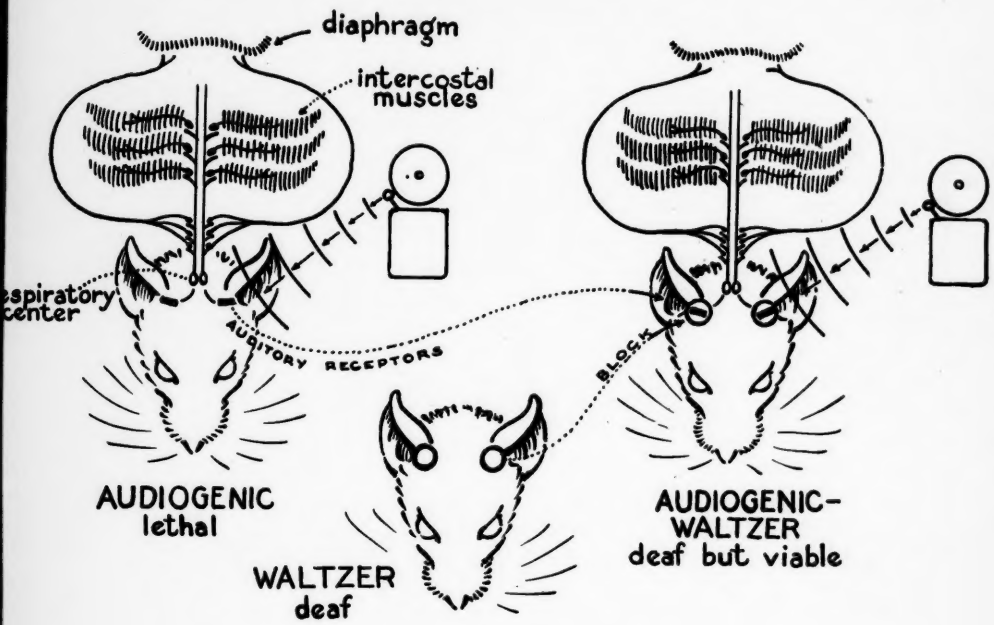

DILUTE BROWN MICE WHO CANNOT HEAR
MAY LIVE TO WALTZ ANOTHER YEAR

(SEE PAGE 296)

Naked-Seed Cotton

Polydactyl Salamanders

Genetics for the Million

Sex-Linked Microphthalmia

No Polyploid Honey Locusts or Partridge Berries

VOLUME 38 OCTOBER, 1947 NUMBER 10

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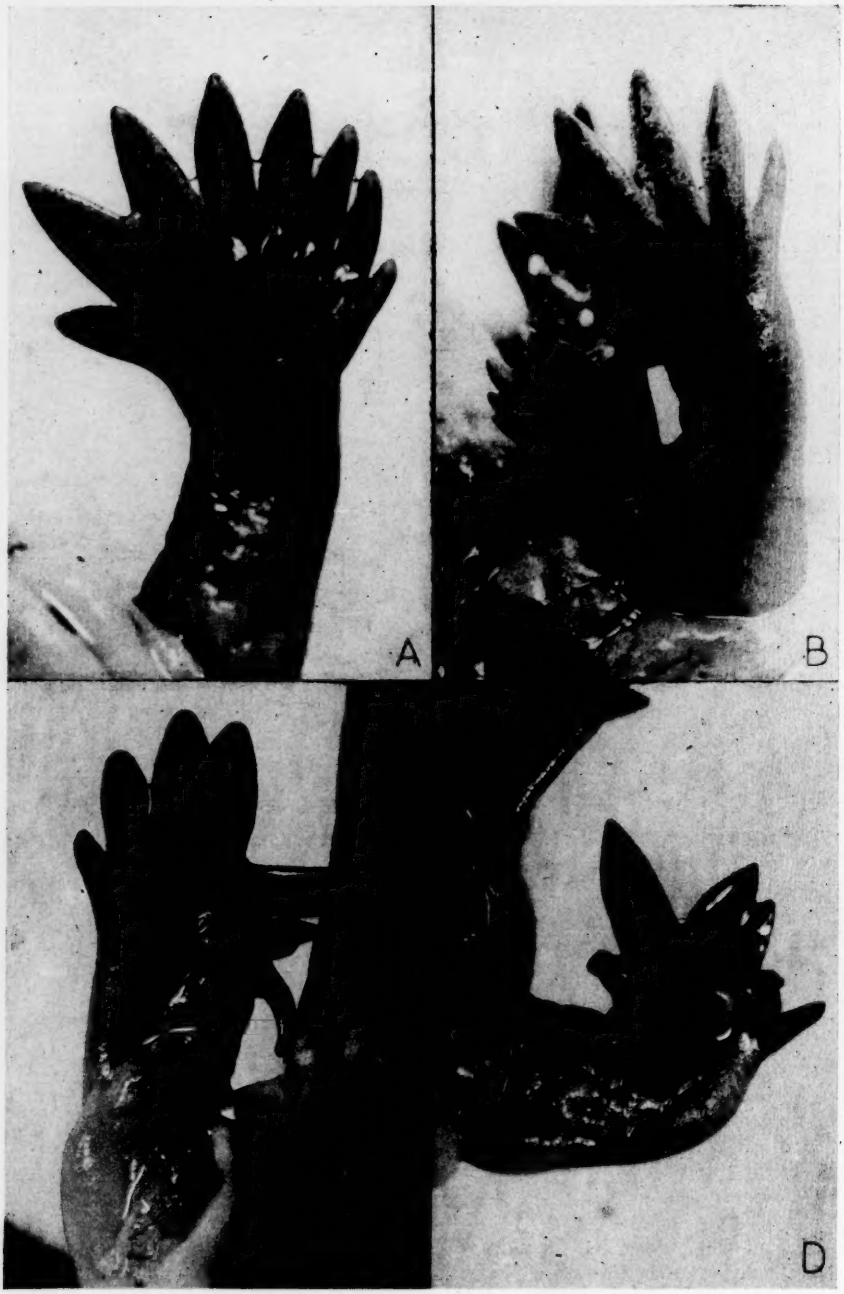

EXTREME TYPES OF POLYDACTYLY IN AMBYSTOMA

Figure 1

A—Eight-toed right hind limb. The first digit is on right. The other hind limb on this animal had the normal five toes. *B*—Supernumerary left limb of another specimen with 12 digits. On the right side of this animal there is another small two-toed extra limb. *C* and *D*—show respectively the left and right hind limbs of an animal in which each foot bears nine digits. Note the addition of toes postaxially and on the palm.

POLYDACTYLY IN THE TIGER SALAMANDER

DAVID W. BISHOP

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POLYDACTYLY is common in domesticated animals and rare cases have been described in feral individuals. Extra digits accompanied by supernumerary limbs occurring on a large scale in a wild population of tiger salamanders is of considerable importance, not only from the genetic point of view but embryologically as well, inasmuch as certain similarities exist with duplications resulting from transplantation experiments.

This report concerns a series of polydactylous salamanders, *Ambystoma tigrinum,* discovered in a high altitude lake near Boulder, Colorado. Seventeen abnormal larvae were collected in October, 1946, and two abnormal adults were found in the spring of 1947 in Muskee Lake, located at an altitude of 8300 feet, in the front range of the Colorado Rockies, about 15 miles west of Boulder. So far as the writer is aware this is the first report of mass polydactyly occurring naturally in *Ambystoma.* Most of the specimens unfortunately had been preserved when received in the laboratory, but four polydactylous animals (two males and two females) were alive and were segregated for breeding purposes in the spring. In spite of injections of anterior pituitary glands and the demonstration of some mating activity, no eggs have been obtained from these animals. It is unfortunate that through a severe winter-kill many possibly polydactylous individuals were lost during the winter of 1946-47. The exact hereditary nature of the polydactylous tendency remains obscure, but considerable evidence points toward the strong influence of a genetic component in the development of these abnormalities.

HEPTADIGITAL PENTAPEDE
Figure 2

Extra left limb with seven digits. The orientation of this supernumerary limb suggests possible dorsoventral mirror-image symmetry.

Nature of the Anomalies

A complete list of the supernumerary digits in the 19 abnormal animals is reported elsewhere.[1] The condition ranges from a single, small extra toe to a multi-digited foot with nine toes. The supernumeraries are usually located postaxially in series with the normal fifth toe, but frequently may be found on the palm or on the back of the foot (Figures 1*C* and *D*). In five cases extra limbs are present, situated in the posterior angle of the normal thigh and the body wall. The supernumerary limbs themselves are polydactylous, having as many as twelve digits, making in one case a total of 18 toes on the combined

POLYDACTYL CLUBFOOT

Figure 3

Adult salamander with six toes on each hind limb giving clubfooted appearance. As is true in all cases observed in this study, the fore-limbs have only the four normal toes.

"normal"-supernumerary limb complex (Figure 1*B*).

The condition is found in both sexes and is present on either one or both sides of the animal, but in the latter case not necessarily in a bilaterally symmetrical pattern. The extra digits first appear in the young nymphs and are carried over into the adult with little or no change (Figure 3).

A significant morphogenetic feature is the fact that the hind limbs only are affected. Among 19 abnormal animals collected in no case is there any indication of polydactyly of the fore-limbs.

The series has been studied carefully for signs of mirror-image symmetry, a common occurrence in experimentally induced duplicates. In only two questionable cases is there any sign of mirror-image symmetry (Figure 2); in the great majority of cases the supernumeraries, both those involving extra digits and those involving extra limbs, are completely asymmetrical.

Discussion

In the absence of direct breeding data the genetic basis for these anomalies

nevertheless is suggested by several lines of evidence.

(a) Among the individuals collected in October, 1946, polydactyly was very common—17 out of 19 collected were abnormal—tending to rule out chance (injury, etc.) as the cause. Moreover this lake and others in the general vicinity, at altitudes up to 10,500 feet, have been extensively collected for salamanders in the past with no signs of polydactyly. The abnormality seems to have appeared in the *Ambystoma* population fairly suddenly.

(b) Extra digits were found in salamanders which developed from eggs laid in two successive years, 1945 and 1946, and while not implying that two generations are involved, this indicates that polydactyly developed in two different seasons and that if exceptional environmental factors played the predominant role, these factors must have been present twice.

(c) The restriction of the anomalies to the hind limbs indicates that some intrinsic factors are involved and that the modification is not merely a chance effect involving limb development in general. If environmental factors alone had induced the abnormalities, as for example sudden temperature or chemical changes, we might expect to find both fore-limbs and hind limbs affected, as both sets of limbs have the same general plan of development, except in point of time. Attention should be called to the polydactylous strain of mice noted by Chase[2] in which the anomaly is limited almost entirely to the hind limbs (personal communication).

(d) Experimentally induced limb duplications in *Ambystoma* generally show mirror-image symmetry following transplantation and this has been interpreted on the basis of the development of more than one growth center followed by reversal of symmetry of one of the centers.[4] Gabriel[3] investigating the effect of colchicine suppression of polydactyly found evidence of mirror-image symmetry in limb duplication in fowl. The general absence of such symmetry in the present series indicates that we are dealing with a different type of development, not one involving a divided growth center and symmetry reversal, but rather perhaps with the continued or repetitive activity of a single growth center under genetic control. In this connection the polydactylous limbs of guinea pigs of Wright[6] and of Scott[5] should be noted in which there is little suggestion of mirror-image symmetry. In their animals polydactyly tends to restore the "lost" digits to the normal limb.

Conclusion

We may tentatively conclude that the polydactyly and limb duplications described here in *Ambstoma* are naturally occurring and have been induced largely through genetic means, the environmental factors playing a relatively small part.

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✦

Genetics Fellowship Announced

The Helen Putnam Fellowship for Advanced Research in the field of genetics or of mental health will be offered by Radcliffe College for the academic year 1948-1949.

This fellowship carrying a stipend of $2,000 for an eleven-month period beginning October 1, 1948, is open to mature women scholars who have their doctorate or equivalent qualifications, and who have research in progress.

Applications should be submitted to Radcliffe College not later than April 1, 1948. The appointment will be announced about May 1, 1948.

GENE THERAPY

CLYDE E. KEELER
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PROBABLY the most common complaint brought by practical-minded medical men against medical genetics is the lack of effective therapies. Hereditary abnormalities are deep-seated in the germplasm. Although the effects of an hereditary deficiency may be relieved temporarily, as by injecting insulin into individuals suffering from diabetes mellitus, this does not purge the germplasm of its defects. Barring elimination of the morbid genes themselves, they are conceived of by the medical man as being fated always to reappear generation after generation, forever.

My studies in the last eight years have led me to the increasing realization that what is in effect a therapeutic technique actually is widely in use in applied genetics, and presumably in nature in the evolution of gene systems. This has the effect of achieving a permanent correction of hereditary diseases or dyscrasis occurring in strains of organisms, by a homozygous modification of the genetic formula. Although at present we may not envisage the application of such gene therapy to medico-genetic diseases in man, it appears worth while to explain the principles on which it operates in plant and animal breeding and presumably in nature. This note describes the selection and manipulation of requisite genes for the therapeutic treatment of hereditarily defective strains.

For purposes of this discussion a hereditary abnormality is considered as any statistically significant deviation from the average (or normal) manifestation of a characteristic determined by genetic factors (genes).

Therapy for abnormal hereditary conditions may be classified as follows:

A. *Environmental Therapy* (always phenotypic)
   1. Supplying of lacking substances
   2. Specialized environment
   3. Blocks (surgical or otherwise)

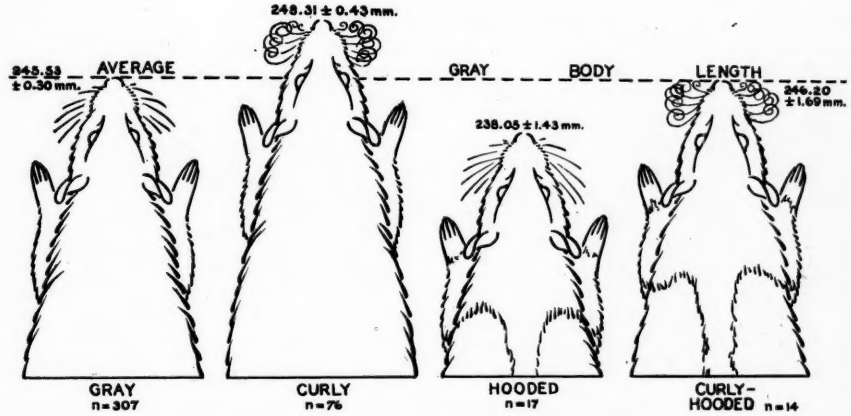
B. *Gene Therapy*
   1. Phenotypic
   2. Heredeo-phenotypic
      a. reciprocal defect cancellation
      b. genetic blocks

In environmental therapy we may provide from other sources the insulin that diabetics cannot produce, the thyroxin in the case of cretinism, or the hydrochloric acid and Castle's intrinsic factor in the case of pernicious anemia.

For those hereditary constitutions that react violently against strawberries, ragweed, fish, and a host of other allergen-bearing substances, we keep the body in perfect health by eliminating these environmental factors from the food or the surroundings. As long as the allergens are kept at a distance, all is reasonably well. Possibly the relationship between allergic tendencies and psychic manifestation may complicate this picture. And it should be borne in mind that the healthy phenotype must forever depend on a controlled environment.

Or, we may provide a particular, specialized environment in which the mutant gene will function in a relatively normal fashion. Thus, environmental therapy of this sort may be demonstrated by the character "reduplicated" in Drosophila, in which increased room temperature lowers the incidence of penetrance, and hence, reduces the proportion of individuals exhibiting the duplicated feet. At 10 degrees the penetrance is reduced by half.

Decreasing the temperature of the skin of Himalayan rabbits[11] and of Siamese cats[13] while the hair is developing will to a great extent correct the "pigment deficiency" that ordinarily gives these breeds a white coat. The moral of this is that Siamese cats can be virtually "cured" of melanism by moving them to the tropics or of "albinism" by transferring them to the Arctic. The cures are permanent as long as the environmental correctives are maintained.



GENIC BALANCING TO CONTROL BODY LENGTH

Figure 4

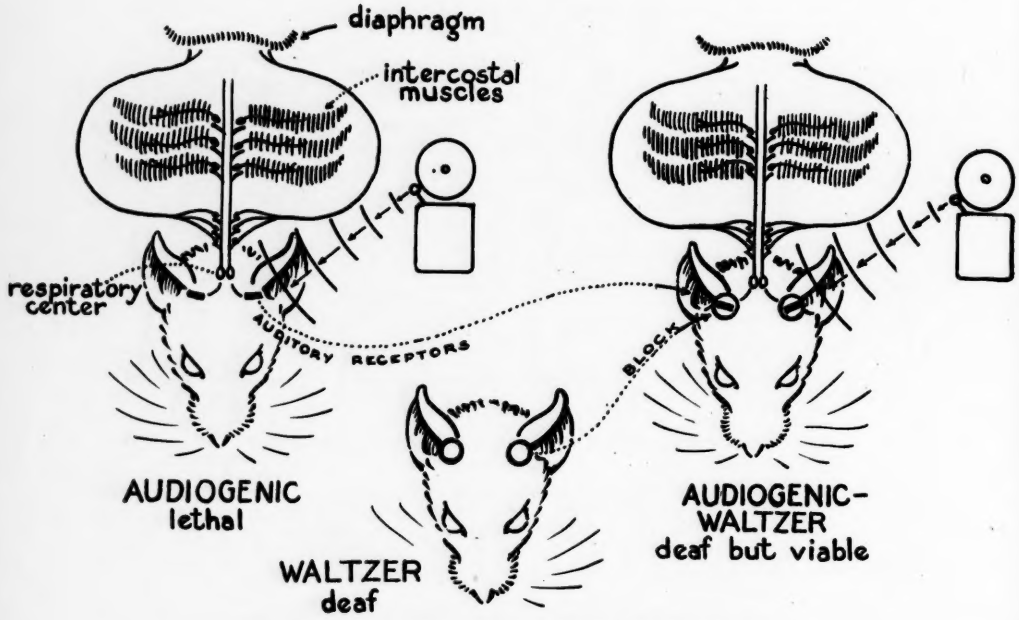
The average gray male Norway rats studied had a body length of 245.53 ± 0.30 mm. The average curly rat exceeded the gray, with a body length of 248.31 ± 0.43 mm. The average hooded rat was inferior to the gray with a body length of 238.05 ± 1.43 mm. When hooded was inserted into curly rats the average curly-hooded animals measured 246.20 ± 1.69 mm., which does not differ statistically from the body length of the gray Norway rat.

Surgically, one may remove defective tissues to prevent more serious consequences that their retention might bring about. The surgical removal of a hereditary pigmented mole to forestall cancer and the prophylactic removal of the spleen in a person predisposed to hereditary hemolytic icterus, are examples.

Phenotypic gene therapy may be demonstrated by size crosses involving multiple factors. The F_1 hybrids between giant and dwarf strains of rabbits[2], for example, are uniform and intermediate in size. Thus, the extreme tendencies in opposite directions cancel each other and produce a normal intermediate. But the F_2 and subsequent generations of such a cross will vary from dwarf to giant so that the therapy is only temporary.

Castle's original study of throwbacks[3] involving merely two genetic loci in the guinea pig is a similar but simplified example of phenotherapy. The red agouti crossed with the black non-agouti guinea pig produced in the F_1 a normal gray guinea pig. (Any F_1 involving a recessive character is a case of gene therapy because the normal dominant gene corrects the alternative abnormal developmental process dependent upon the recessive allel so that the F_1 is normal.) In Castle's case of the throwback containing red and non-agouti, the combination of red and non-agouti genes corrects the individual only so long as it is heterozygous. Individuals homozygous for both red and black are a dirty buff color and hence much more deficient in pigmentation than either red or black. To make a stock homozygous for these characters only makes matters worse.

The above described types of gene therapy are only temporary and must be applied to the individuals of each generation. But there is another type of gene therapy (heredeo-phenotypic therapy) that cures the strain as well as the individual, with the result that this therapy permanently cures or readjusts the germplasm. This is seen in nature in situations in which adverse genes are "rectified" by the piling up of modifiers to reduce the dominance of the morbid genes. This concept, first pointed out by R. A. Fisher[6], has been found operative in many situations. In the laboratory it is a common observation that the first



BLOCKING AUDIOGENIC SEIZURES GENETICALLY

Figure 5

Mice of the dilute brown Jackson Laboratory strain (*dba*) die in convulsions when a bell is rung. Theoretically, the insertion of the waltzer (deaf) character into audiogenic strains should block the lethal nervous pathway and thus save the lives of mice exposed to fatal audiogenic stimuli.

time a particular gene mutation arises, the specimen bearing it is a weakling. If it is possible to nurse the mutants to adulthood and get them to breed, the mutant stock eventually derived several generations later is greatly improved in viability and health.

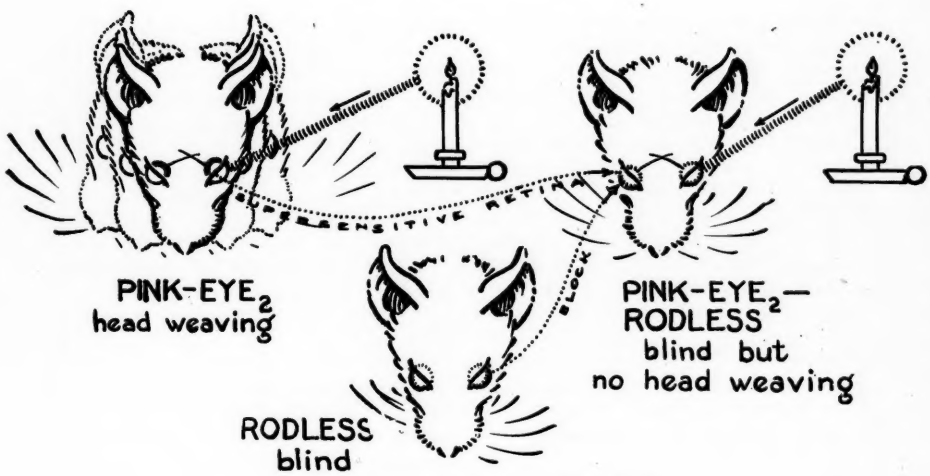
I recall that "hairless" in rabbits and "stub" in rats were weaklings to start with, but increased in viability after a few generations. Cases of this sort appear to be frequent in Drosophila.*

Snell informs me that the waltzers of today at the Jackson Laboratory are not nearly as active as were their ancestors years ago, i.e., the "waltzing" instability is undergoing modifications in the direction of the normal. Cloudman feels that the "mosaic" and "spotty" strains have improved in viability. A dwarf mutation in mink appears to have increased in viability over several generations.[10]

I have shown that it is possible to correct permanently several statistically significant gene-conditioned deviations from the normal in certain mutant coat-character strains of the Norway rat.[9]

Thus, when the curly gene is introduced into the Norway rat in the homozygous state (see Figure 4) the animal is statistically larger[8] than normal in body length and body weight (a tend-

*After this paper was accepted for publication there appeared a note by Dobzhansky and Spassky[5] demonstrating that x-ray induced mutations will correct the reduced viability of multiple-mutant Drosophila stocks. These authors state: "To detect beneficial mutations, strains that carry deleterious genetic variants, which reduce the viability below normal, must be chosen as materials; in such strains, mutations that cancel deleterious effects and bring the vigor and viability back to normal may be expected to occur."



A DRASTIC CURE FOR HEADWEAVING

Figure 6

Pink-eye$_2$ mice suffer from head-weaving as a secondary compensation for nystagmus. This results in confusion and affects the mice adversely. Rodless (blind) mice are well adjusted since mice normally employ their nasal sense much more than their visual faculties. Theoretically, the insertion of the rodless gene into a pink-eye$_2$ stock wil lcorrect the confusion of pink-eye$_2$ by blocking the hypersensitive pathway.

ency to gigantism). Conversely, when the hooded gene is introduced into a normal strain, the body size (length and weight) is reduced (a tendency to nanism). But if we want to correct permanently the gigantism effect produced by the curly gene, without sacrificing the curly hair characteristics, we may do so by the gene therapy of adding hooded in homozygous form to the homozygous curly strain. Thus, the purebred curly hooded strain is permanently corrected for size deviations, because the curly strain is thereafter genetically purebred also for hooded.

We may take a similar example from the study of curly and ruby-eyed dilute (r.e.d.) strains of rats. As we have said above, curly strains are statistically above the average in size. R.e.d. animals are below the average in size (both length and weight). Now instead of employing hooded to correct the gigantism produced by the curly gene, we may use r.e.d. The resulting curly-r.e.d. strain will remain normal in size.

Here should be mentioned the size effects associated with blue and short ears genes in the house mouse studied by Castle et al.[4] Larger size than normal was associated with blue dilution. Smaller size than normal was associated with short ears. Animals homozygous for both blue and short ears were normal in size. As in the rat examples, a tendency to larger size is permanently corrected by adding a tendency to smaller size.

If we want to correct the greater length of the curly rat, but make it into a proportionately stockier animal, we can do so by adding the waltzing gene.

It may be an advantage to be able to choose between a number of different genes in order to find the most appropriate correcting factor, that injures the animal least in other respects. Thus, hooded animals in general may possibly tend to be more irritable and not quite as healthy as r.e.d. animals. I do not have, however, any controlled physiological or psychological observations upon the combinations of hooded with curly.

The above therapy might be designated as the *reciprocal defect concella-*

tion method. Sometimes the "cure" may initiate other morbid reactions. The cutting of the facial nerves to prevent neuralgia by blocking nerve impulses, may result in facial paralysis. Similarly the result of gene therapy may be to damage the organism in other ways.

Surgeons think nothing of enucleating the eyeball in cases of inherited glioma retinae to prevent death, or of excising portions of the rectum to stay off demise by cancer of that organ arising from inherited polypi, or surgically removing the hereditarily deformed appendix[12] to prevent the dangers inherent in appendicitis. This therapeutic blocking has its counterpart in certain genetic procedures, of which a few examples may be given.

Headweaving, (presumably the compensation of a nystagmus tendency) is found in a low percentage of pink-eye$_2$ (Roberts) mice. This constitutes a visually confusing characteristic among these animals, and one might even suggest that since mice depend upon their sense of smell much more than they do upon their eyes, it would be to their advantage to sacrifice their vision. If we are willing to pay the price of vision, a gene against head weaving in the form of the rodless character may be inserted into the pink-eye$_2$ animals curing the confusion of head weaving (see Figure 6) but depriving these individuals of sight. Animals homozygous for the correction will breed true for the correction as well as for pink-eye$_2$.

Recently, Hall[7] reported the presence of audiogenic seizures in dilute brown mice. This characteristic is found in a high percentage of the dilute brown strain. The characteristic may be fatal in the unusual environment of a ringing bell which sets off a series of lethal seizures. Theoretically (see Figure 5) we could block the lethal nerve pathway from ear to brain by adding the homozygous waltzing gene to deafen the animals.

Incidentally, this example is an instance of a detrimental gene that in a specialized environment suddenly takes on survival value by adapting the organism to an otherwise lethal situation. Of course, this conclusion is based on the moot assumption that it is better to be a deaf mouse than a dead one. Cases of modifiers removing the lethal effects of a gene probably occur in nature.

In the same fashion hereditary dwarfism that produces sterility might be alleviated to some extent by breeding in the gene for "pug"[1]. The dwarfism is caused by a deficient pituitary; the "pug" (acromegaly) by an overactive pituitary.

As applied to the psychological field, wild, excitable temperament may be cured by the breeding in of tameness genes as I have shown to have been done in the "taming" of the laboratory albino rat.[9] Here the additive effects of the genes for black and hooded and to a lesser extent, albinism, may be observed.

The savage nature of the dominant Bluefrost gene in mink is moderated by the addition of a pair of recessive Silverblue genes.[10] One could say as correctly that the abnormal tameness of Silverblue mink is partially corrected by the addition of the Blue-frost genes.

Thus, it would appear that gene therapy may be applied to three levels of genetic deviations: physical, physiological and behavioristic and that the keeping in mind of the principles of gene therapy may be of advantage in solving a number of practical genetic problems.

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GENETICS FOR THE MILLION

An Unfinished Story

THIS first Pelican Book* on genetics is clearly and entertainingly written. It should appeal to thousands as a rewarding and painless enlivenment of the dull bus ride between office and home. Where the talk is of the genes, or of the individual and his relation to his own heredity, the story moves clearly and the few errors are trivial. The concept of races or other groups differing mainly in gene frequency, so that their means are genuinely different although the individuals overlap, is made about as clear as it could well be in a book written to read easily.

But when the authors come to fit the facts of genetics together to show John Q. Busrider how these facts affect race and society and what could or should be done about it, important errors occur. These are likely to engender in John Q. a pessimistic or hostile attitude toward eugenics, and they may leave his thinking about individual and racial differences dangerously muddled. There is a tendency to deprecate the importance of heredity and to intermingle biased social and economic theories with established biological facts.

Pessimism about Eugenics

By confining their discussion exclusively to negative eugenics, the authors make it easy for the unwary reader to take a jaundiced and pessimistic view of what eugenics is and what it might accomplish. The authors' dim view of "eugenics" is best given in their own words summing up the chapter on this subject, pages 82 and 83: "If this [i.e. allowing defectives to reproduce] is allowed to go on for thousands of generations without countermeasures, the situation might become serious indeed. But, fortunately, this dismal prospect is as yet too remote for us to worry about. The hue and cry about the coming biological suicide of mankind is, to say the least, premature." And on page 73 discussing albinism, they say: [If all albinos were sterilized] "It would take about two hundred generations (some 5,000 years!) to reduce the proportion of albinos in the population to half the present frequency."

Their skepticism is based on a detailed consideration of the example they choose as the type case, the fate of a rare recessive defect so serious that it must inevitably have been selected against in precivilized cultures. They then compute what will happen to the frequency of such a gene if selection against it ceases. This assumes that unfavorable mutation is the only cause of genetic deterioration in mankind. Added to this is the pessimistic way of phrasing the results of negative eugenics. Taking their cue from Zeno, they consider the number of generations required to halve the frequency of a defective gene rather than the gain per generation, thus tempting John Q. to conclude that the Achilles of selection never can catch the tortoise of recessive defect. The natural rarity of each such defect is emphasized. But it is not pointed out in this context that there are *many hundreds* of different kinds of rare recessive defects. True, this is mentioned in another context on page 61: "Indeed over a hundred different hereditary eye diseases are known, some dominant, others recessive." Does the general reader toward whom this book is beamed have the background to see the relation of these facts?

What Selection Can Do

Let's look at this point in some detail. If one per cent of individuals in a population are homozygous recessive for a certain gene and if all reproduction of

*DUNN, L. C., and THEODOSIUS DOBZHANSKY. *Heredity, Race and Society.* Pp. 115. Price 25 cents. Penguin Books, Inc., New York, 1947. (The Pelican books are paper-covered "Penguin" books on scientific and technical subjects.)

such affected individuals were prevented, it would require a bit more than four generations to halve the frequency of occurrence of that defect. Sounds pessimistic, doesn't it? Another way to appraise the same data is to say that one generation of preventing the reproduction of such affected individuals would reduce the frequency from 1 in 100 to 1 in 121. Thus the very first generation of such selection would remedy about one-sixth of the trouble. This is still a rebuke to and a debunking of those who imagine that such selection would instantly be completely effective, but it is less pessimistic than implying that centuries must elapse before *any* good would result!

Here we come to the nub of the practical problem: how balance the gains against the cost, and how evaluate each? To put the story more specifically in human but conservative terms may help to reach an intelligent judgment. If a serious monogenic recessive defect is already so rare that only one defective is born among 10,000 normals, then the prevention of reproduction by all such individuals would lower the frequency of the defect in the next generation by about one fiftieth. If there were 500 different defective genes (and there may be more*), nearly five per cent of all individuals born will be defective in one way or another. That would be roughly 1½ million grossly defective individuals born in the United States in each generation from this cause alone. Would preventing the birth of one-fiftieth of these unfortunates be worth while? The 1,470,000 who would be born anyhow are certainly a part of the picture and are bound to be disappointing to those naive souls who infer that a generation of complete prevention of reproduction by undesirables would clear up *all* the trouble. But what would it be worth to reduce the load of defect by 30,000? In cash and tears do the gains balance the costs?

The authors point out that sterilization of affected individuals would eliminate bad dominant genes in a generation, and if it were possible to detect carriers of recessive genes a sterilization program could be really effective. This they believe will eventually be possible. But even if it were, they hold that such a program would be ill advised. "Finally let us not forget that treatments may and probably will be found that will cancel the manifestations of hereditary defect, as insulin relieves the manifestation of hereditary diabetes." Whether the way to racial good health lies in the direction of medical corrections for defective genes might be debated. The prognosis in diabetes is not always rosy even with insulin universally available.

Also the assumption of random mating in man is not quite fulfilled. Mating is measurably assortive for continuously distributed characteristics like I.Q.,† stature, and many others. This increases the variation and permits a given amount of selection to be somewhat more effective than if mating were wholly random. The amount of inbreeding in man is too small to make much difference when the frequency of an undesired recessive is moderately high but, as the writers point out, few of the genes against which selection has long been practiced can be in that class. The frequency of the undesired recessive individuals will be $(1\text{-}q)^2 + Fq(1\text{-}q)$, where F is the coefficient of correlation between uniting egg and sperm for likeness with respect to a single pair of genes. Geography, propinquity, and racial, religious and social preferences in the marriage choice, may well give F a value of as much as .01 to .05 in many human populations (Cf. Dahlberg's emphasis on the "isolates" in Sweden). When recessive individuals occur at the moderately high rate of 1

*In the United States about one live birth in 170 is physically malformed. In addition, and not detected at birth there are about two percent mental defectives—and a good many physical defectives will show up later. At 2 million live births per year; that is about 50 thousand defectives born per year, or 1½ million per generation. Not all of these defects are genetic, of course, but many are.

†HALPERIN, S. L. *Jour. Ment. Def.* 53.153-163. 1946.

in 100, a value of .01 for F means that the undesired genes are being eliminated about one-tenth faster than under the Hardy equilibrium equation on which the authors base their computations. But if the defectives are as rare as one in 10,000, then even as low an F-value as .01 would mean that the elimination of undesired genes proceeds almost twice as fast as they state.

The possible effectiveness of negative eugenics is limited, to be sure, and has often been grossly exaggerated by overzealous enthusiasts in the past. But will this discussion really clarify the picture? Thousands of John Q's are likely to conclude that it is as clear as day that consideration of eugenics can safely be postponed a few centuries without risking more than a trivial decline. Indeed this debatable position has already been made the basis for an article* which will be read by many thousands who will not see the complete version in the Pelican book, and who are deprived of the chance to evaluate the total argument. What if the conclusions there so "boldly affirmed and plausibly maintained" are in fact untrue?

Possibility of Negative Selection

Even though the "biological suicide" of mankind may still be remote, his cultural suicide may be quite near at hand, a possibility the authors do not consider. They rest their argument on the assumption that selection may cease, entirely, but do not go on to ask what will happen if it actually turns negative. The existence of differential birthrates in man and their possibly adverse effect on qualities which, from our viewpoint, seem desirable, is not discussed. Yet this factor seems to be hundreds of times as powerful in shifting gene frequency as is the slow increase in frequency of harmful genes by mutation, on which alone the authors rest their argument. Probably the major genetic hazard to continued cultural progress lies in the fact that mankind has now created a "civilized environment" in which many genes with detrimental effects on the maintenance or improvement of that civilization have acquired positive selection values.

The area in which such selective forces impinge most critically on society is in the effect they may have on the decline of intelligence. This is a subject regarding which there have been a plethora of biased and alarmist statements. But the stubborn fact remains that every careful and conservative study of the subject finds significant evidence that present birth differentials portend a continuing erosion of genes for general intelligence. Lorimer and Osborn, and the Population Committee of the National Resources Board† have recently surveyed the situation in the United States.

*The Case Against Sterilization. *Science Digest*, February 1947. Pp. 75-80.

†Lorimer, Frank, and Frederick, Osborn. ***Dynamics of Population.*** Macmillan, 1934. ***Population in a Changing World.*** Population Committee of the National Resources Board. Government Printing Office. Washington. 1938. Lorimer (private communication received after this was in press), commenting on the Lorimer-Osborn estimate, points out that in census and survey data which furnish the basis for such estimates it is not possible to isolate in a "pure state" the cultural-environmental factors from the genetic factors whose interaction produces the end result measured by the I. Q. test or other estimate of intelligence. In response to a question as to limits, Lorimer considered that the correlation between expressed intelligence and genetic endowment must certainly be positive and hardly less than 0.5. This appears conservative as compared with Dunn and Dobzhansky's summary of the evidence on mental ability as adjudged by intelligence tests of identical twins: "Here 65 to 80 per cent of the differences are due to heredity" (p. *22*). Since the situations are not the same, the agreement between these estimates must be quite close. It would obviously be misleading to allege that I. Q. differentials in social and regional studies are *exclusively* to be attributed to genetic differences, just as it would be to claim that they are exclusively an artifact of the environment. The existence of a gene-erosive trend appears to be definitely established even though room remains for argument (and, more important, for research) to determine the rate at which the trend is altering the constitution of the gene-pool of the population. Only if the above correlation were practically zero could we dismiss birthrate differentials as having no significance.

The former authors consider the reality of the decline to be a "moral certainty," and place it between .89 and 4.4 I.Q. points per generation. More recently the situation in England has been examined by Thompson* and by Burt† apparently with the intent to get at the facts carefully and cautiously, regardless of whether they are pleasant or not. These recent British studies indicate that the average I.Q. is declining at least one point, and possibly more than two points per generation, solely because of the negative correlation between I.Q. of child and number of sibs. This lies within the rate bracketed by Lorimer and Osborn for the U. S. population. The remarkable unanimity as to the reality and the adverse affects of birth differentials suggest that these estimates must lie within gunshot of the truth. The trend is unmistakable, and important.

This decline is more ominous than might appear superficially. The I.Q. scale was initially constructed so that the population average would be near 100. When the I.Q. falls below 90 the individual approaches a level at which he ceases to function effectively in a complex world. A decline of one or two per cent per generation may sound trivial but actually it means that the population is using up in each generation something like one-tenth to one-fifth of the margin between functional efficiency and the disaster of intellectual blackout. To be sure, a population averaging only 90 in I.Q. will still contain many individuals above 100 and even a few above 120, but the reservoir of ability needs to be large enough so that at least the minimum necessary number of competent leaders and managers get elected, hired, appointed or otherwise put at the controls of manufacture, production, transportation, government, and the other activities essential to maintaining the present kind of civilization. Not far from an average I.Q. of 90 there must be a level below which there just are not enough such competent individuals to man the controls of our complex civilization. This point varies with the complexity of the civilization and with the accuracy of the mechanism by which the man and the job are brought together. Burt considers that the present British trend will mean that "in a little over 50 years the number of pupils of 'scholarship' ability would be approximately halved and the number of feebleminded almost doubled."

Obviously these are dangers we will not have to wait 5,000 years to detect. Since the authors recognize that differences in mental ability are "65 to 80 per cent due to heredity" (p. 22), and since they elsewhere mention genes for "normal mentality" it is clear that they would not agree with the Watsonian dictum that the I.Q. has no genetic component. Apparently they merely forgot to relate the genetic facts to the facts regarding differential birthrates cited above; yet here are social factors of crucial importance to the maintenance of a democratic way of life. An intelligent population may not have a democratic form of government, but it is hard to imagine that an increasingly unintelligent population will maintain a democratic tradition. A feebleminded population begs for the domination of a dictator. Here heredity and society touch very intimately; and yet this whole matter is passed over without comment.

Reliance on Mass Selection

The authors have understated the practical utility of considering relatives, and in some relevant contexts even race, when estimating what an individual will be or do.

On page 110 we read: "The personal qualities of an individual are more important than the race from which he

*Thompson, G. "The Trend of National Intelligence." *Eugenics Review,* April 1946. (To be published in an expanded book form in 1947.)

†Burt, Cyril. *Intelligence and Fertility.* Survey prepared at the request of the Royal Commission of Population. Pp. 43. Price two shillings. The Eugenics Society and Hamish Hamilton Medical Books. London 1946.

came." And on page 104 we find, in connection with the blood groups and blood transfusions, that: "the old and obsolete theory of heredity and the ideas about race based upon it would counsel you that a blood most similar to yours would be found in a person of the same race, and particularly in your close relatives, brothers or sisters." "But you had better disregard such advice." And a little later we read: "It is wiser to choose your donor according to his blood type, which is determined by his individual heredity, than according to the race from which he sprang. It is this property of his blood that matters, not his skin color, intelligence, or morals."

True, there is increasingly less need for a pedigree estimate as the individual's own phenotype can be more accurately appraised. For the blood types the accuracy of identifying the individual phenotypes is well-nigh perfect and the process is simple and quick. Hence there is little practical need to consider relatives and race at all in this particular case, save only in emergencies where the test cannot be run. Moreover the utility of considering race is rather low even then, because the frequencies of the four blood groups vary only moderately as between most races. The point the authors stress that compatible bloods cut right across racial lines, is of course, sound and in need of emphasis.

Nevertheless, there are many characteristics not so accurately measurable in the individual as the blood groups. When accuracy in measuring the individual phenotype is lacking, the characteristics of relatives automatically become more useful for prediction purposes. By treating an extreme case as if it were typical the authors have led John Q. to have too much confidence in mass selection and too little in the general usefulness of genetic methods as bases for predicting what an individual will be or do.

The Variability of Races

Several lances are broken on "the refuted 'blood' theory of heredity" but the reviewers were puzzled all the way from page 32 to page 108 about what that theory was supposed to be. At least two refuted theories are involved: that blood, the physical substance, is the vehicle by which inheritance is transmitted (pp. 31 & 32); then it becomes the idea that inheritance is a blending process and is verbally scourged accordingly. This confusion detracts from an otherwise clear section on "ancestral heredity" (pp. 47-49). The emphasis is so strong as again to invite the inference that considerations of pedigrees have no real meaning or usefulness. This is mainly because purely verbal arguments so easily suggest all-or-none conclusions.

The authors have done a good job of explaining that differences between races are largely differences in gene frequency, rather than all-or-none contrasts which are present in every member of one race and are absent from all members of another, and that purity of race does not mean complete uniformity and is not synonymous with homozygosity. But the wording is often equivocal enough to imply some wrong conclusions about the variability of populations. Some instances have already been cited. Here are a few more:

On page 101 we read: "The hereditary diversity of a group, be it a family, a clan, or a race, persists indefinitely." Assuredly the forces tending to extinguish intra-group genetic variance are feeble, except when inbreeding is intense, but *they are not zero*. To apply to "a family, a clan, or a race" the Mendelian algebra of *infinite* populations, as the authors do, is to overlook one of the potent causes of genetic differentiation between groups.

Also on page 101 we read "the diversity, the variation, found in a race is more important than the racial averages." If that means what it appears to, it says that the intra-racial variance is *always* larger than the variance between racial means. Did the authors really intend that? Isn't it true of some characteristics, especially when the races being considered do not differ widely, but untrue of other characteristics, even in the same

population, and more especially in populations where the races are very diverse?

Near the bottom of page 112 we read: "If all peoples on earth were to intermarry at random, the resulting humanity would not, as is frequently but mistakenly supposed, be some kind of a compromise between all the now existing races. It would rather be an extremely variable lot: some persons resembling each of the now existing races would continue to be born, but other individuals would have combinations of traits that are rare or nonexistent at present." Both authors have had enough experience with crosses between lines or races differing in many genes to know that this simply does not occur. If the mean of a parent race differs from the mean of the F_2 generation by much more than three times the F_2 standard deviation, the parental types will not be recovered among the F_2 segregates in such a finite population as can occur in a world like ours. This is on a par with telling a novice at bridge that 13-spade hands will be encountered.

The same error appears on page 114 where it is stated of race crossing leading to race fusion that "Such a course would lead merely to interracial variability being replaced by individual variability." In truth the increase in individual variability would not be nearly enough to replace entirely the lost interracial variability. Under the simple conditions for which Castle proposed his formula for number of loci involved in quantitive inheritance, only $\frac{1}{2N}$ of the initial individual variance which was due to differences between racial means in a population consisting of equal numbers of two different races would reappear in the individual variance within F_2 or subsequent generations produced by panmixia. Most of the possible exceptions to the conditions Castle specified would operate to make the actual fraction even smaller. Thus Muller showed (*Amer. Nat.* 70:409-442) how certain—probably rare—conditions of gene frequency and dominance make it possible for an F_2 actually to be less variable than either of the parent races which were crossed to produce it.

Emotionally-tagged Words and Ideological Biases

The account is unduly defensive and often deprecates the importance of heredity wherever it is verbal, although nearly always fair and unbiased wherever the presentation is wholly quantitative.

Some examples are: (1) On pages 5 and 6 are listed some characteristics which—"are determined *very largely* by heredity . . ." (italics ours) and then for contrast are listed some which ". . . are determined not by heredity, but by where we happened to be born, . . ." The absence of any qualification like the "very largely" when the second group is mentioned, especially when that group includes "the diseases we get," illustrates the tendency to minimize the importance of heredity wherever it must be admitted to be a major factor, and by innuendo to make it easy for the reader to suppose heredity has *no effect at all* wherever its effects are less important than those of environment.

There is at least a little argument by abuse of the motives of those who do not agree with the authors and by extolling the motives of those who do. Galton is misquoted on page 8: "Galton was convinced that the English propertied and governing classes were a repository of virtually all that is precious in the English nation and possibly in mankind," whereas in Galton's own words which they quote we find "eminent" but not "propertied" or "governing"! They are not the same thing. On page 10 we read: "It is not surprising that writers *with a desire* to create such categories should have been *swayed by prejudice and self-interest.*"

Elsewhere ideological biases and value judgments are allowed to appear as if they were facts as well demonstrated as such biological realities as blood groups, taste blindness and skin pigmentation. On pages 6 and 7 World War

II is portrayed as a struggle between "the democratic solution" and "a totalitarian solution" to the problem of human differences. John Q. would get from this no hint that communism and fascism operate substantially alike, as far as concerns individual liberty to behave according to those human differences, with the democratic ideal being altogether unlike either of them in this respect. On page 10 we read: "Powers over human life as great as those required to put a eugenical program into effect can be entrusted *only* to a society in which *social and economic equality* of individual and groups is guaranteed and protected in positive ways." (Italics ours.) It seems odd that authors who have just spent so much time explaining the facts of physical and biological *inequality* are so sure that it is necessary to fit everyone to the Procrustean bed of social and economic equality!

Here again the implication is strong that eugenics is about 90 per cent compulsion. Says Frederick Osborn, "A system of arbitrary control would not be eugenics, it would be simply the application of genetic science to the breeding of specific kinds of men and women. . . . Democracy is therefore the necessary safeguard of eugenics."

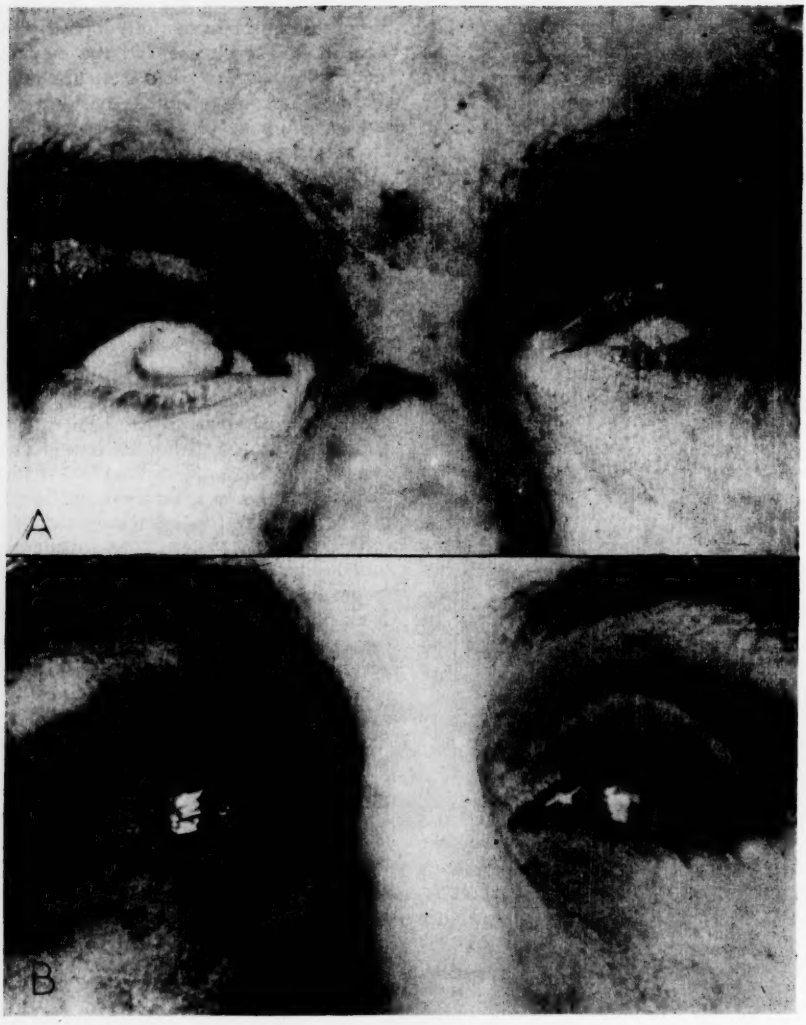
The Final Score

Do the biases and errors in emphasis we have discussed in detail deserve the space accorded them? These reviewers are convinced that a very important issue is at stake. To them the evidence seems clear that the simply fantastic changes in the human environment in the past century have altered greatly the forces and the checks and the controls that have ruthlessly but actually guided human evolution in the past. Effective and humane ways must be found to replace these controls by education and voluntary cooperation rather than by coercion. This is too urgent a matter to ignore or let go by default. Galton was right that heredity is important in human society. His idea that "forces under social control" could alter heredity is confirmed by the record: look at shifting birth rates. Up to now these portentous forces have been acting blindly.

The problem remains, even though a great deal of nonsense has been spoken and written about eugenics. Neither Barzun nor Madison Grant have clarified the problem by exaggeration, but it cannot be removed by ignoring it or dismissed because fanatics get queer notions. The democratic way seems to be to get out the truth: half-truths give us Nazi racism on one hand and Soviet Lysenkoism on the other. Something is seriously wrong when geneticists seem to have to forswear their discipline when ever human genes are concerned.

Chapter I started out like a home run, but it seems to these reviewers that the authors were tagged out somewhere near second base while they stopped to discuss some social or economic theories, to placate an ardent environmentalist or to make a face at the late Madison Grant.

Robert Cook
Jay L. Lush



SEX-LINKED BLINDNESS

Figure 7

Appearance of eyes of blind microphthalmic brothers (V-2 and V-4 in pedigree, Figure 8). This defect has been transmitted by normal females for four generations in a Utah family.

A CASE OF SEX-LINKED MICROPHTHALMIA

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MICROPHTHALMIA is a term applied to a wide variety of conditions characterized by small eyes wherein the vision is subnormal or absent. The eyes may be small and the socket normal or the eyes and socket may both be small. The eyes may vary in size from nearly normal to minute. There seems to be no adequate standard by which to classify microphthalmic eyes. Julia Bell concluded that there are no clearly defined criteria as to just what constitutes this condition. Several cases of this defect have been reported showing different kinds of inheritance. It is quite possible that they represent several distinct mutations all classified as microphthalmia.

The present investigation deals with a form of microphthalmia found in four generations of a Utah family. The affected individuals were all born blind and remained so throughout their lives. The pedigree chart (Figure 8) shows the occurrence of the character. In all, seven persons in this kindred are known to have been blind. Only two of them are living at the present time. Reliable information regarding the others has been obtained from members of the family. One of the blind boys, V-4, has been examined by a competent ophthalmologist† and his case diagnosed as microphthalmia. Figures 7*A* and *B* show the nature of the defect and the similarity between the two brothers, V-2 and V-4.

Review of Literature

Julia Bell[2] after analyzing a collected series of cases of microphthalmia, concluded that either sex may transmit the defect. She emphasized, however, the need of examining individual pedigrees. Macklin[8] reported seven pedigrees in which microphthalmia was inherited as a recessive trait, the parents in each case being normal. Usher[12] described a case where this same defect acted as a dominant trait, and in 1922 Ash[1] reported a case where the type of inheritance was a sex-linked recessive. Roberts[11] extended this study. He showed an association between microphthalmia and mental deficiency. The different cases of microphthalmia varied greatly, as did their association with mental deficiency. In two cases there was no mental deficiency, but in no instance in a family was there a case of mental deficiency without microphthalmia. Cases of hereditary microphthalmia have been found in rats and fowls as well as in man. Helen King[7] was unable to obtain a pure strain of microphthalmia by crossing rats in a strain showing the defect. Jeffrey[6] found bilateral microphthalmia in fowls inherited as a simple recessive trait.

Description of Family

In the pedigree chart shown in Figure 8, I-1 and I-2 were residents of England. Of their nine childen one son, II-10, was blind. The oldest daughter, II-3, was born in 1839. She and her sister, II-6, came to the United States and settled in Utah. She became the polygamous wife of II-2, who had two sons and three daughters by his first wife. Four of these children married and had families (not shown in pedigree). All descendents from this marriage have had normal vision. It is through his second

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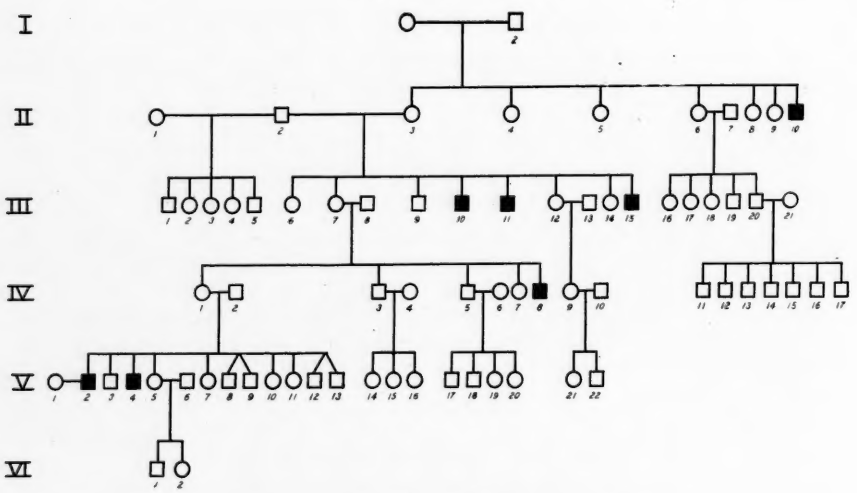
†Diagnosis was made by Dr. Bascom W. Palmer.



FOUR GENERATIONS OF MICROPHTHALMIA

Figure 8

The first generation were residents of England. The oldest daughter was born in 1839. With her sister II-6 she came to Utah as a young woman. She has transmitted microphthalmic blindness to her sons, a grandson, and two great grandsons. Half of her great granddaughters in the affected sibship in generation V would be expected to carry microphthalmia.

wife, II-3, that blindness is transmitted. She was the mother of eight children, four girls and four boys. Three of the four boys were born blind and never married. Two daughters of II-3, III-7 and III-12, married and had families. III-6 and III-9 died in infancy and III-14 had no children. III-7 had two daughters and three sons. One of the sons, IV-8, was blind. III-12 had one daughter and two grandchildren, all of whom were normal. IV-3 married and had three daughters, all with normal vision. IV-5 married and had four normal children, two sons and two daughters. IV-7 had no children. The oldest daughter of III-7, IV-1, married and had eleven children, seven sons and four daughters. Two of the sons, V-2 and V-4, were born blind. These two boys are living at the present time. V-2 is a salesman who goes from house to house selling merchandise directly to the customers. A Seeing-Eye dog assists him in his rounds. He married but has no children. His wife was partially blind from diabetes and died soon after their marriage. His eyes are smaller than normal, the left one being smaller than the right. The cornea is covered with a white opaque tisue which prevents anything but a superficial examination of the eye. The eye can be rotated in the socket but there is some difficulty in opening the eyelid. The size of the eye socket and the palpebral fissures seem to be normal. The ptosis of the eyelid may be due to the smallness of the eyeball and to the lack of use of the eyelid.

V-4 has a bachelor of science degree and is now doing graduate work at the University of Utah. He shows special ability in mechanical skill and repairs radios, weaving machines, bicycles, etc. His eyes are very similar to his brother's, as can be seen from the photographs (Figures 7*A* and *B*). Like his brother's, his eyes are covered with white opaque tissue which prevents examination of the inner eye. The left eye is also smaller and the eyelids can be opened with less difficulty than in the case of

his brother. The eye socket and palpebral fissures are normal in size. He can rotate his eyes but has no control over the movements, having no idea as to which way they are rotating.

The other sister who came from England, II-6, left her husband and all of her six children with the exception of one son, III-20, in England. There were three daughters and one other son. Nothing is known, however, about any of them or of their children except that one of the daughters spent about a year in this country. The children of III-20 all had normal vision. Five of them died in infancy.

Inheritance

Since this blindness occurs only in males in four different generations and is transmitted only by normal mothers to their sons, it appears to be a sex-linked recesive trait. II-3, III-7 and IV-1 are apparently all carriers of the defective gene. While there is no evidence that the other females who married are carriers, they have not had enough sons definitely to exclude this possibility.

Eugenic Significance

It would appear that in the family of IV-1 blindness may show up in the sons of any of the three living daughters. V-5 is married and has one normal son and one normal daughter. V-7 is dead and V-10 and V-11 are eighteen and sixteen years old respectively. This condition involves a serious eugenic problem in these three girls. Each of them has an even chance of being a carrier of the defective gene since their mother carries the gene. If either or both of them should be carriers each of their sons would have an equal chance of being blind and each daughter an equal chance of being a carrier like the mother. On the basis of the pedigree data, each son born to any of these girls has one chance in four of being blind. Obviously it would be very important to determine whether or not these daughters are carriers of the defective gene. In an effort to determine this, the mother's eyes were examined to see if there were any character in common with the eyes of the two blind boys which might possibly serve to identify the heterozygous condition. The daughters' eyes were also compared with the mother's in a search for significant resemblances. Nothing significant was found. The mother's eyes were large and apparently ophthalmologically normal. No trait could be detected which would suggest that she might be heterozygous.

Other Heritable Traits

An effort was then made to find other hereditary traits in the sons and mother which might be linked to the gene for blindness and possibly serve as an indicator of the presence of the defective gene in the normal daughters. Both blind boys have short stubby thumbs. This condition is found in but one of the mother's thumbs. It is also found in one other brother, V-3, who has normal vision. The mother, the grandmother and the two sons are also afflicted with varicose veins. If either of these traits were linked with blindness they would also be sex-linked and they might be used as possible indicators of whether or not the daughters were carriers.

Stubby thumbs are usually inherited as a simple autosomal dominant and there is no reason to believe that they are inherited differently in this family. While varicose veins occur here as if they might be due to a sex-linked dominant gene, they occur so frequently in the general population and there are so few cases involved in this family that the chances of their being inherited as a sex-linked dominant is remote. However, the three daughters will be watched with interest for the development of this trait.

Summary

Cases of microphthalmia occurring in four different generations of a Utah family are reported. They occurred only in males and the inheritance seems to be that of a sex-linked recessive. Since some of the females are potential carriers of the defective gene, some of the eugenic implications are discussed.

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◆

THAT SETTLES IT!

Dr. Curt Stern,
% The Journal of Heredity,
1507 M Street, N. W.,
Washington 5, D. C.

Dear Dr. Stern:

I have read with the greatest interest your article in the August 1947 issue of the Journal of Heredity, "The Skin Color of Children from White by Near-White Marriages," in which you suggest the improbability of a black child being born to two apparently white parents. In the course of your article you ask the question, "will a dark child result from the union of two apparently white parents, one of whom has negro ancestry?" The answer to this question will be quite apparent to you when you read the following facts which both a prominent physician and a well-known criminal investigator vouch for.

John Hebron of Atlanta, Georgia, a light-skinned negro, married a white woman, Effie ——— (who later married Grant Munro, an Englishman). The issue of John and Effie was a coal-black negress, Lucy Hebron. Effie said of her first husband, that "a nobler man never walked the earth. I cut myself off from my race in order to wed him, but never once while he lived did I for an instant regret it. It was our misfortune that our only child took after his people rather than mine. It is often so in such matches, *and little Lucy is darker far than her father ever was.*" There, Sir, is the answer to your question. The distinguished physician who reported this case in detail was none other than John E. Watson, M.D., and the well-known detective who was in charge of the investigation was Sherlock Holmes Esq., of 221B Baker Street, London, England.

For a further account of this interesting case I would refer you to page 402 of *The Complete Sherlock Holmes* (Garden City Publishing Co.) where the case is reported as *The Yellow Face.* The name A. Conan Doyle (who was in reality a literary agent for Holmes and Watson) is erroneously given as the author of this volume.

Very sincerely,

William L. Hires

Box 44
Ardmore, Pa.

CYTOGEOGRAPHY OF GLEDITSIA AND MITCHELLA

EARLENE ATCHISON

The Blandy Experimental Farm, Boyce, Virginia

THIRTY collections* of *Gleditsia triacanthos* L., involving both armed and unarmed (f. *inermis* (Pursh.) Fassett) types, and eighteen collections of *Mitchella repens* L. have been cytologically examined for evidence of intraspecific polyploidy. *G. triacanthos,* the honey locust, extends naturally from the Appalachians to central Kansas, from southern Ontario and Wisconsin to Georgia and eastern Texas; the collections listed in Table I more or less cover this range. *M. repens,* the partridge berry, has a discontinuous distribution throughout the states east of the Mississippi River and extends north to Nova Scotia; only Appalachian and eastern coastal plain collections are included in the table. Both species have been subjected, in the course of geological time, to the changes in climate and topography to which various intraspecific polyploid races have previously been attributed.[1,3,4]

Chromosome counts of *G. triacanthos* and *M. repens* (Figure 9) were obtained from young leaves pretreated with paradichlorobenzene[5] and smeared in acetic orcein. No polyploid individuals were found in either species. *G. triacanthos* was consistently found to have a chromosome number of $2n = 28$; *M. repens,* $2n = 22$.

Large-leaved, large-fruited collections of *M. repens* were obtained from various localities in North and South Carolina (Table I). These specimens maintained their gigas characters after transplanting in Charlottesville, Virginia. Similar individuals were later found to be fairly frequent among populations of the species along the eastern coastal plain. Their apparent independence of

TABLE I. Cytogeographic distribution of *Gleditsia triacanthos* L. and *Mitchella repens* L.

State	County	Place and Collector*
Alabama	Dallas	Selma—**1**
		Orville—**1**, 1
Arkansas		Hot Springs—**11**
Florida	Leon	Tallahassee—**17**
	Santa Rosa	Pensacola—**20**
Georgia	Raburn	Clayton—**9**
Indiana	Jackson	Vallonia—**14**
	Tippecanoe	Lafayette—**23**†
Kentucky		Cumberland—**5**
	Caldwell	Princeton—12
Minnesota	Winona	Winona—**4**
N. Hampshire	Cheshire	Dublin—22
New York	Tompkins	7
N. Carolina	Cumberland	Fayetteville—21, 18-6‡
		Hope Mills—1
		Pierce Mill—21
	Harnett	Byrds Mill—**1**
	Orange	Durham—**1**
Ohio	Franklin	**13**§
Oklahoma	Woodward	Woodward—**19, 10**
		Mooreland—**10**
S. Carolina	Horry	Myrtle Beach—1§§
Tennessee	Knox	Norris—**3**#
	Anderson	Clinton—**3**
Texas	Nacogdoches	Nacogdoches—**15**
	Harrison	Marshall—**15**
	Angelina	Lufkin—**8**
Virginia	Clarke	Boyce—**1**
	Albemarle	Charlottesville—**21**
	Pr. Edward	Hampden Sidney—**1**
	Mecklenburg	Clarkesville—**1**
	Charlotte	Keysville—2
	Rockingham	Elkton—21
	James City	Williamsburg—21
	Albemarle	Afton—21
	Clarke	2
W. Virginia	Hampshire	Romney—2
	Grant	Gormania—2
	Lewis	Weston—**16**

* The numbers refer to the collectors listed below. A number in bold face (**1**) indicates *G. triacanthos.* A number in ordinary type indicates *M. repens.*

1. Earlene Atchison
2. J. T. Baldwin
3. L. F. Bailey
4. T. K. Bergh
5. H. L. Borden
6. M. F. Buell
7. R. T. Clausen
8. M. V. Dunmire
9. V. B. Elliott
10. R. B. Gierow
11. C. X. Grano
12. A. M. Harvill
13. L. F. Kellog
14. C. Locke
15. E. R. Mueller
16. R. H. Quick
17. R. O. Schornhurst
18. B. W. Smith
19. J. E. Smith
20. P. B. Tabor
21. O. E. White
22. M. E. Whittemore
23. J. W. Wright

† Three collections.

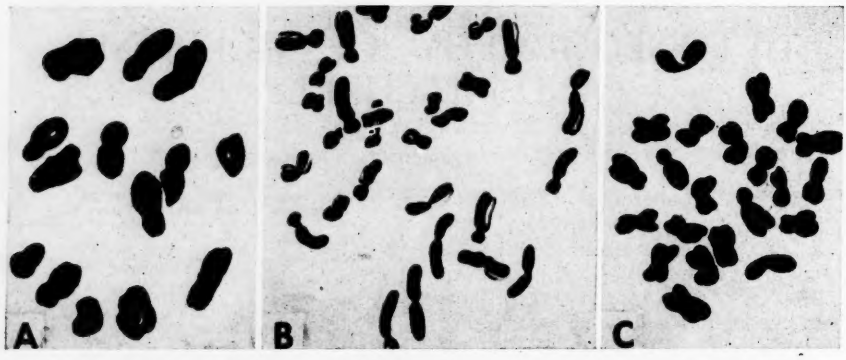
‡ Three collections, two of which were large leaved.

§ Two collections.

§§ Two collections, one of which was large leaved and fruited.

Two collections, one "Millwood Honey Locust," the other "Goldworth."

*The author wishes to acknowledge her indebtedness to the many individuals who collected seeds and plants for this study. See footnote to Table I.



CHROMOSOMES OF HONEY LOCUST AND OF PARTRIDGE BERRY
Figure 9

A—Metaphase, first meiotic division of chromosomes of *G. triacanthos* showing 14 pairs of chromosomes. *B*—Somatic metaphase of same showing 28 chromosomes (3350 ×). *C*—Somatic metaphase of *M. repens* showing eleven pairs of chromosomes (5000 ×).

ecological surroundings suggests that the gigas plants are mutant forms.

Baldwin,[1,3] Baldwin and Culp[4] in cytogeographic analyses of various North American plants have suggested that polyploid races may often result from climatic and topographical change, and thus are probably a characteristic of many species in the Appalachian range. Intraspecific races were demonstrated in *Galax,*[1] *Sedum ternatum,*[3] *Diospryos virginiana;*[4] races cannot yet be demonstrated in *Oxydendrum arboreum,*[2] *G. triacanthos* or *M. repens.* Initiation and fixation of polyploid races, or species, implies not only the presence of environmental stimulants but also a selective advantage of the polyploid derivative. Intraspecific polyploidy, therefore, appears to be dependent on the plasticity of the genotype of individual species, and is probably not a common phenomenon.

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✦

Random Mating, Chain-Letter Style

This chain was started in Reno in the hope of bringing happiness to all. Unlike most chains, this one does not cost you money. Send a copy of this letter to five of your male friends, then bundle up your wife and send her to the person that heads the list. When your name works up to the top of the list you will receive 15,175 women.

Have faith. Do not break the chain. Let the good work go on.

Note.—One man broke the chain and got his own wife back!

—Anonymous airmail received at this office.—Ed.

A RECESSIVE NAKED-SEED CHARACTER IN UPLAND COTTON

J. O. Ware, L. I. Benedict and W. H. Rolfe*

SEEDS of Upland cotton (*Gossypium hirsutum* L.) are usually characterized by a covering of adherent fuzz that remains attached over the entire coat after the longer lint has been removed by ginning. There is, however, a wide variation in the intensity and extent of this fuzz. Some varieties have a very heavy and intense covering while others display varying patterns of thinness, sparseness, tuftedness or even of no fuzz at all. Even when no fuzz is present the seeds may not be entirely naked, because varying quantities of thin white felted hairs remain attached to the black seed coat after ginning. These hairs represent the basal segments of the lint broken off in ginning. This lint residue is distinct from the true fuzz, and is correlated with the amount of lint present on the seeds. When the seeds of a variety are entirely devoid of lint, the lint bases are altogether absent and the seed is completely naked. Such "no lint" varieties are known.

The senior author has made a study of these variations and set up standards for classifying the degree of seed-fuzz in a series of 20 grades.[5] This grading scale has been used throughout the experiments described here. He has summarized genetic work previously done on some of the variants, and showed in this and other work that the naked seed character found previously in upland stocks had in each case been dominant over fuzzy seeds or the partial-fuzz condition.[5, 6, 7]

The present writers have recently found a recessive naked seed character in a non-commercial upland variety having brown lint. This variety presumably has been maintained intact and in isolation from regular commercial stocks since its introduction from a foreign source in Colonial times. Overby[3] has given an account of this cotton, the details of its preservation as a separate stock and its use in home manufacture by the French Acadians of the Teche country of Louisiana. Ware[8] has designated this stock "Acadian Brown" (Figure 10, left).

Materials and Methods

Seeds of Acadian Brown obtained through John R. Cotton of the University of Louisiana were planted at Florence, S. C., in the spring of 1938. It was crossed that season with eight fuzzy seed varieties (see footnote, Table I) and with one naked seed variety, Acala Mex, in which the naked character is dominant. The Acala Mex variety was secured for planting at Florence, S. C., in 1937 from the Sacaton, Arizona, collection of Dr. T. H. Kearney. This variety originated from a variant Upland plant collected by Kearney and associates near Hermosillo, Sonora, Mexico, in 1924.

In the summer of 1938 the Acala Mex also was crossed with nine fuzzy seed varieties and reciprocally crossed with Acadian Brown. Five of the fuzzy seed varieties were among those with which Acadian Brown was crossed and the other four were additional varieties (see full list in footnote to Table II).

The fuzzy seed varieties were self pollinated lines which had been checked for purity as to reproduction of the character. In the course of the early part

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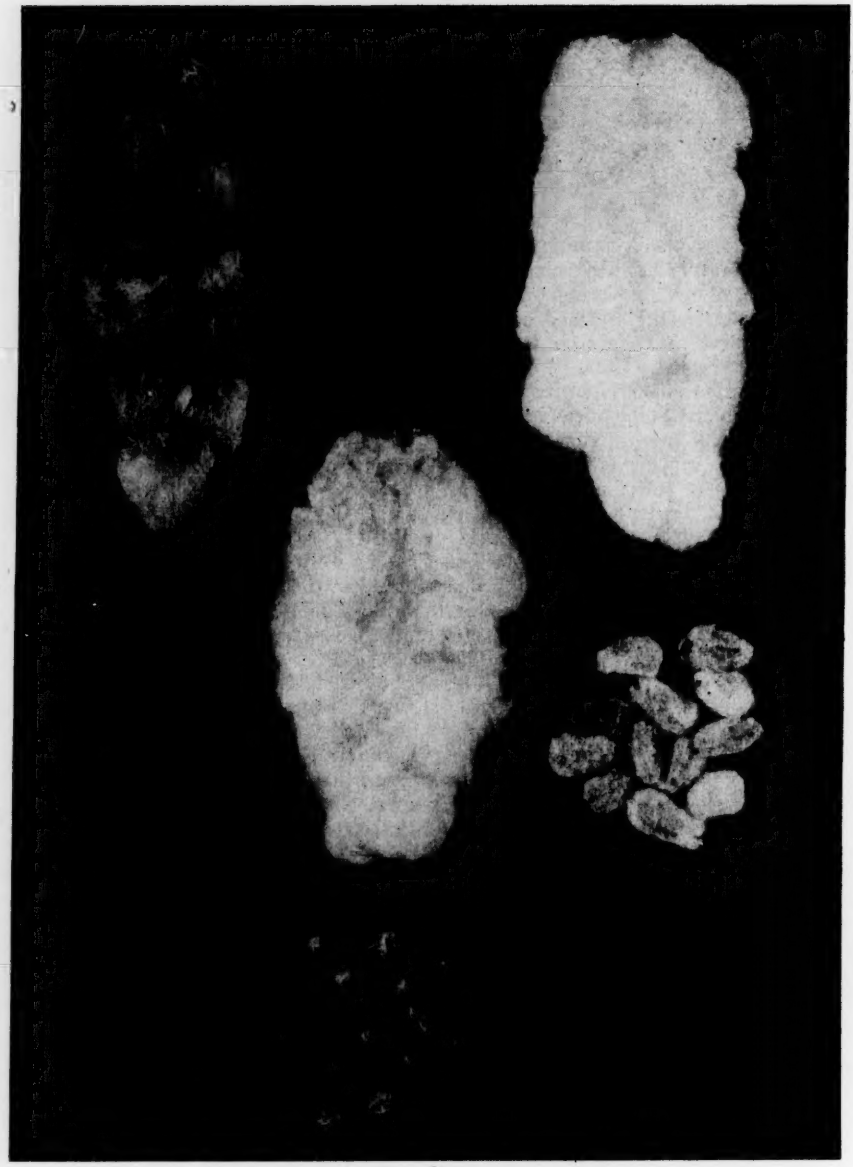


LOCKS AND SEEDS OF PARENT TYPES

Figure 10

Left, Acadian Brown, seed grade 15; *right,* Half and Half, seed grade 3; *lower center,* F_1 seed (grade 4) showing recessiveness of the Acadian Brown naked seed in this cross. In all other instances of crosses between naked and fuzzy seed cotton, the naked character has been dominant.

of this work the Acadian Brown and Acala Mex were likewise checked for stability of their naked seed expressions. This verification was completed by the end of the F_1 generation which was grown in 1939. On finding by examination of the material in the winter of 1939-40 that the Acadian Brown naked seed character was recessive, the purity of the parent plants used in the crosses could be checked by the nature of the seed of their progenies. The 1939 season was the third year of growth in this experiment and the second year of self pollination for Acala Mex. Two years of selection and selfing was sufficient to check the purity of this stock for the dominant naked character. The two naked seed varieties had not been previously exposed to each other under open pollinated conditions. This checkup indicated that all Acadian Brown and Acala Mex plants used in the 1938 crosses were pure for the characters under study in this experiment.

In the crossing of Acadian Brown and the eight fuzzy seed varieties, pollen was taken from one plant each of the latter group and respectively crossed with one or more separate Acadian Brown plants. Eight respective fuzzy seed and twenty Acadian Brown plants were involved (see list of fuzzy varieties and crossings in the footnote to Table I).

All plants included in the crosses were also self-pollinated to produce progenies for verification of purity for the character and for comparison with the F_1 generations. Before the selfed and crossed seed were prepared for the 1939 planting, the seed grades of the parents involved were determined. As indicated in the footnotes of Table I, all fuzzy seed plants were of grade 3 except the Rowden sample, which was grade 2. All Acadian Brown plants were either of grades 14 or 15. The product of the 1939 growth, that is, the eight fuzzy and twenty Acadian Brown progenies, and the 20 F_1 groups, were each graded plant by plant for seed cover. These data are summarized in Table I.

The same procedure was followed in the crossing of Acala Mex and the nine fuzzy seed varieties, *i.e.*, pollen was taken from one plant each of the latter group and respectively crossed with one or more separate Acala Mex plants. Nine respective fuzzy seed and 22 separate Acala Mex plants were involved as detailed in Table II. As in the other set of crosses the plants included were self-pollinated, and before the 1939 plots were planted, all the seed was graded. The grades of the fuzzy plants ranged from grade 2 to grade 5 inclusive, and the grades of the Acala Mex plants were either grade 15 or 16 (see footnote Table II). The grades of the progenies from these plants, as with the Acadian

TABLE I.—Recessiveness of naked seed in F_1. Summary of crosses of Acadian Brown with eight fuzzy seed varieties.

Varieties and First Cross	Population number and frequency of F_1 and progeny of selfed parent plants by classes and grades														
		Fuzzy class								Naked class					
	Number	2	3	4	5	6	7	8	9	13	14	15	16	17	18
Acadian Brown[1]	285									7	81	132	50	9	6
Fuzzy varieties[2]	115	19	96												
F_1[3]	326	1	88	66	38	48	71	12	2						

[1] Twenty plants, 7 of grade 14 and 13 of grade 15, separately planted and progenies combined in frequency distribution in naked class.

[2] Eight plants, one each of Rowden, Florida Green Seed, Half and Half, Hastings Upright, Brownseed Rowden, Round Leaf, Brymer Brown and Red Okra Nankeen separately planted and progenies combined in frequency distribution in fuzzy class. The Rowden plant was of grade 2 and the other seven of grade 3.

[3] Twenty separate crosses made between plants of following descriptions:
One Rowden, grade 2 × three Acadian Brown, grade 15 plants
One Florida Green Seed, grade 3 × two Acadian Brown, grade 15 plants
One Half and Half, grade 3 × four Acadian Brown, grade 14 plants
One Hastings Upright, grade 3 × two Acadian Brown, grade 15 plants
One Brownseed Rowden, grade 3 × three Acadian Brown, grade 15 plants
One Round Leaf, grade 3 × two Acadian Brown, grade 15 plants
One Brymer Brown, grade 3 × one Acadian Brown, grade 15 plant
One Red Okra Nankeen, grade 3 × three Acadian Brown, grade 14 plants
The 20 F_1 growths of these crosses combined in frequency distribution in fuzzy class. This position in the chart shows recessiveness of the fuzzy seed character. The cross of Half and Half × Acadian Brown is illustrated in Figure 10.

Brown group of crosses, were also determined and the summary data are shown in Table II.

Reciprocal crosses, as previously mentioned, were made in 1938 between Acadian Brown and Acala Mex. Pollen was taken from one Acadian Brown plant and crossed on two Acala Mex plants and likewise pollen was taken from another Acala Mex plant and crossed on two additional Acadian Brown plants. The six plants involved were also self-pollinated and after maturity the seed was graded. The three Acadian Brown plants fall in grade 14 and the three Acala Mex plants in grade 15. In 1939 offspring of these plants were grown in six separate progeny rows and the four lots of crossed seed in other respective rows. On grading the seed of this growth the plants were combined into three frequencies: one for Acadian Brown, one for Acala Mex and one for the F_1 (see first three lines Table III).

Pressure of other work forced the abandonment of this study until the spring of 1944. Then the seed of three F_1 plants of one of the Acadian Brown × Acala Mex crosses (grade 15) were planted along with check-rows from seed of two Acadian Brown plants (grade 14) and of two Acala Mex plants (grade 15). Seed classification of the F_2 and parental line plants were made the following winter. These data are shown in the last three lines of Table III. The recessive condition of Acadian Brown naked seed is illustrated in Figure 10, and factor interaction resulting in fuzzy seed from the cross of the two naked seed varieties, Acadian Brown and Acala Mex, is shown in Figure 11.

TABLE II.—Dominance of naked seed in F_1. Summary of crosses of Acala Mex with nine fuzzy seed varieties.

Varieties and First Cross	Population number and frequency of F_1 and progeny of selfed parent plants by classes and grades									
		Fuzzy class				Naked class				
	Number	2	3	4	5	13	14	15	16	17
Acala Mex[1]	331					—	20	191	97	23
Fuzzy varieties[2]	132	15	87	15	15					
F_1[3]	240	—	—	—	—	53	123	62	2	—

[1] Twenty-two, 14 of grade 15 and 8 of grade 16, separately planted and progenies combined in frequency distribution in naked class.

[2] Nine plants, one each of Half and Half, Hastings Upright, Brownseed Rowden, Round Leaf, Red Okra Nankeen, Higginbotham Brown, Arvin Brown, Red Okra Green, and Algerian Brown, separately planted and progenies combined in frequency distribution in fuzzy closs. First six plants grade 3, Arvin Brown, grade 2; Red Okra Green, grade 4; and Algerian Brown, grade 5. In the frequency distribution, all plants in grade 2 from Arvin Brown, all in grade 4 from Red Okra Green, and all in grade 5 from Algerian Brown.

[3] Twenty-two separate crosses made between plants of following description:
One Half and Half, grade 3 × one Acala Mex, grade 15 plant
One Hastings Upright, grade 3 × three Acala Mex, grade 15 plants
One Brownseed Rowden, grade 3 × two Acala Mex, grade 15 plants
One Round Leaf, grade 3 × one Acala Mex, grade 15 plant
One Red Okra Nankeen, grade 3 × one Acala Mex, grade 16 plant
One Higginbotham Brown, grade 3 × three Acala Mex, grade 16 plants
One Arvin Brown, grade 2 × three Acala Mex, grade 15 plants
One Red Okra Green, grade 4 × four Acala Mex, grade 15 plants
One Algerian Brown, grade 5 × two Acala Mex, grade 16 plants
The 22 F_1 growths of these crosses combined in frequency distribution in naked class. This position in the chart shows dominance of the naked seed character.

Results and Discussion

The summary of the F_1's and parent lines of the crossing study of Acadian Brown with the eight fuzzy seed varieties, in Table I, shows naked seed of the Acadian Brown to be recessive to the fuzzy seed of these other varieties, but that dominance of the fuzzy character is incomplete. The 285 plants of the combined Acadian Brown lines ranged from grade 13 to grade 18 inclusive, they were all definitely naked. The variation in

Table III.—Interaction of naked seed characters from Acadian Brown and Acala Mex in F_2 of cross and summary comparison of parent progenies, F_1 and F_2.

Varieties F_1 and F_2	Population number and frequency of progeny of selfed parent plants, F_1 and F_2 by classes and grades										
		Fuzzy class			Naked class						
	Number	4	5	6	13	14	15	16	17	18	19	20
Acadian Brown[1]	78	—	—	—	2	50	22	2	1	1	—	—
Acala Mex[2]	74	—	—	—	—	4	38	20	12	—	—	—
F_1[3]	78	—	—	—	—	15	38	25	—	—	—	—
F_2[4]	49	3	—	5	—	12	10	5	4	2	5	3
Acadian Brown[5]	32	—	—	—	—	27	5	—	—	—	—	—
Acala Mex[6]	23	—	—	—	—	4	19	—	—	—	—	—

[1] Three plants each grade 14 planted and produced the 78 progeny plants in 1939.

[2] Three plants each grade 15 planted and produced the 74 progeny plants in 1939.

[3] Four lots of crossed seed planted and produced the 78 F_1 plants in 1939.

[4] Three F_1 plants each grade 15 planted and produced the 49 F_2 plants in 1944. Photograph of the F_2 seed grade segregates shown in Figure 11.

[5] Two plants each grade 14 planted and produced the 32 progeny plants in 1944.

[6] Two plants each grade 15 planted and produced the 23 progeny plants in 1944.

feltedness which causes a spread of five grades corresponds to the amounts of lint the particular plants bore. The variation in lint index or lint percentage in this group of plants was considerable.

The 115 plants of the combined eight fuzzy seed lines were rather uniform for fuzz grade, a few being in grade 2 and most of them in grade 3. On the other hand, the 326 plants of the twenty crosses (see footnote 3 of the table) ranged from grade 2 to grade 9 inclusive, or nearly all the way across the fuzzy grade scale.* Eighty-nine of the F_1 plants were as fuzzy as the parents, but the other 237 spread among the other six grades, the series having a pattern of progressively less and less fuzz. In Figure 10 an example of one of the crosses is shown. The random samples shown in this photograph have the following grades: Acadian Brown, grade 15; Half and Half, grade 3; and the F_1, grade 4. The samples of the parents represent the mode in the frequency distributions of the parent lines of Table I. The sample of the F_1, however, is somewhat above the mid-point of the F_1 population spread shown in this table. Both the data in Table I and the illustration in Figure 10 demonstrate clearly that the naked seed character from Acadian Brown is recessive, but that the fuzz seed character of the several varieties is not completely dominant.

The summary in Table II shows that the naked seed character from Acala Mex, as in all previous work in upland cotton[5,6,7] is completely dominant to the fuzzy character. It will be noted in the table that the ranges of the 331 Acala Mex and the 240 F_1 plants are both within the limits of the naked class range. The shift of the F_1 to grades 13-16 as compared with the Acala Mex range of grades 14-17 can be attributed to increased feltedness resulting from the increase of broken-off basal lint segments. Quantity of lint from the fuzzy parents being dominant to a relatively high degree, the lint index or lint percentage was much higher in the F_1 than in the Acala Mex parent lines. The plants of the fuzzy parent lines all fell in grade 3 except those from three varieties: Arvin Brown (grade 2), Red Okra Green (grade 4) and Algerian Brown (grade 5). The data in Table II definitely confirm previous work that the naked seed character of Acala Mex is dominant.

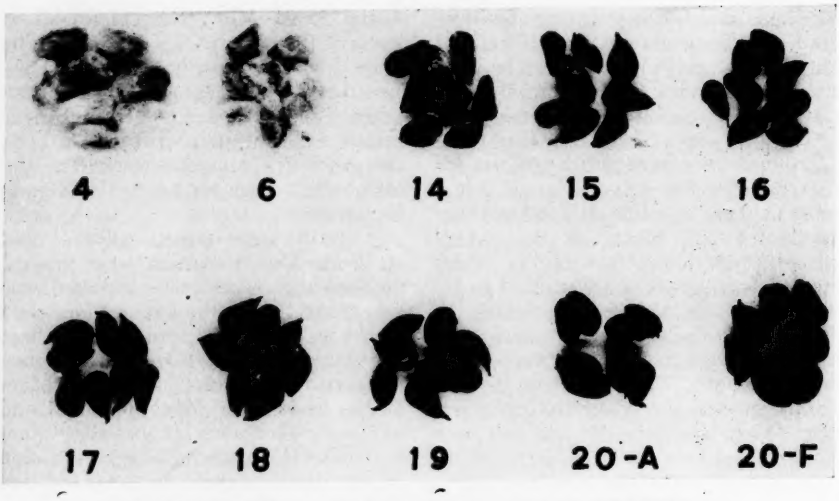
When the seeds of the Acadian Brown $\times$ Acala Mex F_1 plants were graded, the seed coat condition, as expected, was naked like that of the parent plants and their progenies. The spread of the three distributions, as well as the frequency in particular grades of the Acadian Brown and Acala Mex progenies, and of the F_1 generation (upper three lines of Table III) is attributable to variation of lint stub feltedness on the black seed coat among the plants so classed.

Since one of the naked genes is dominant and the other recessive, the two being independently inherited, interaction was expected in the F_2 (fourth line of Table III). This occurred and the segregation of the population spread across a range of 17 of the possible 20 grades. There was some discontinuity in the distribution, however, the plants falling into only nine grades. Only grades 4 and 6 were in the fuzzy class and the other seven grades (14 to 20) inclusive were in the naked class. The nine grades, showing grade 20 as two subgrades, 20A and 20F, are illustrated in Figure 11. In the interaction to the fuzzy class the eight plants classed as fuzzy did not display as much fuzz as is characteristic in most upland commercial varieties (averaging about grade 3), but sufficient to demonstrate the expected interaction.

The lint amount level or percentage also interacted considerably in this F_2 population. Data on the lint percentage are not presented in this paper, but it was much higher in the fuzzy segregates than in any of the fuzz-free segregates.

The lint index also varied greatly within the naked class, becoming lower as the grade number increased. This

*The entire scale, including grades 1 and 10, is shown in an illustration in citation 5.



RANGE OF SEED COAT COLOR IN THE F_2 PROGENY
Figure 11

Seed-cover grades observed in F_2 segregation in hybrid of Acadian Brown × Acala Mex. Note that there is a clear dichotomy between the fuzzy segregates (grades 4 and 6) and the naked segregates (grades 14 to 20). The distribution observed in F_2 was 13 naked:3 fuzzy, due to the combined action of a dominant gene for naked seed and an independently inherited recessive gene for naked seed.

condition is indicated by the range of lint stub feltedness shown on the black seed coat. In grade 19 there was little lint and in grade 20 none whatever. In the full absence of lint the seed tend to grow together or adhere (Kidney cotton). This condition is illustrated in grade 20A in Figure 11, and is of common occurrence in upland cotton whenever the mutant is practically devoid of lint.

Segregation in F_2

The Acadian Brown and Acala Mex progenies summarized in the last two lines of Table III were grown in 1944 along with the F_2 for comparison in the same season. Their grade ranges are somewhat less than in previous years and possibly due to a more steady quantitative modifier level controlling the lint brought about by further selection in grades 14 and 15 respectively (see parent plant grades footnotes Table III). As mentioned above, the feltedness of the naked seed, in general indicates the amount of lint that has been removed by the gin.

The F_2 segregation, 41 naked:8 fuzzy, fits closely into a 13:3 modified dihybrid ratio reported in F_2 crosses between certain white breeds of poultry. Hadley[1,2] obtained a colored phenotype in the F_2 of a cross between the White Leghorn (dominant white) and the White Plymouth Rock (recessive white). In the F_2 the two classes, the white birds numbered 110 and the barred or dark birds numbered 18. According to calculation by the writers these numbers did not depart significantly from the 13:3 ratio (χ^2 1.85, P between .10 and .20). As explained by Hadley[1,2] the White Leghorn has a gene for color, but its expression is prevented by epistatic action of a dominant inhibitor (*I*). In segregates homozygous for the recessive allele *i*, the birds show the color. The white character in the White Plymouth Rock on the other hand is a true recessive. Sinnott and Dunn[4] mention that

the 13:3 ratio also occurs in the F_2 of the White Leghorn and the White Wyndotte. The white character in the latter and in a few other white breeds also is recessive.

The 13:3 ratio in the Acadian Brown × Acala Mex F_2 indicates that the dominant naked seed character in upland is the result of epistatic behavior of an inhibitor rather than a simple dominant allele of a fuzzy gene. This form of naked seed therefore is genetically fuzzy, but the expression is hidden by the epistasis of an inhibitor. The recessive naked character of the Acadian Brown on the other hand appears to be a simple recessive like that of the recessive white in the White Plymouth Rock and White Wyndotte breeds of poultry.

The F_2 interaction of the two naked seed characters in upland may be illustrated by combinations of the letters "I" for inhibitor or epistatic action, "r" for recessive naked, "i" for absence of inhibitor, and "R" for fuzzy which is expressed when "I" is replaced by "i". The combinations and results are shown in the following diagram:

P	Acala Mex *II RR* (naked)	×	Acadian Brown *ii rr* (naked)			
F_1	*Ii Rr* (naked)					
	Combinations	Expected		Phenotype	Observed	Calculated
F_2	*II RR*	1	13	Naked	41	39.81
	II Rr	2				
	Ii RR	2				
	Ii Rr	4				
	II rr	1				
	Ii rr	2				
	ii rr	1				
	ii RR	1	3	Fuzzy	8	9.19
	ii Rr	2				

The phenotypic frequency, 41 naked and 8 fuzzy, as compared with the calculated numbers in the above diagram show little deviation, therefore, giving a good fit to the postulated 13:3 ratio ($\chi^2 = 0.187$, P between .60 and .70).

The dominant or inhibitor type of naked seed has been the only kind of naked seed character in Upland heretofore reported. This form of mutant has spontaneously occurred in practically all of the Upland varieties and appears rather frequently in some of these varieties. Since this variant is characterized by a rather low lint percentage it is rouged out of seed stock lines or strains when its occurrence is rare. However, when the mutant becomes more frequent, as has been the case in some stocks, the whole lot is discarded and another stock substituted. Naked seed, among commercial growers, is commonly accepted as a marker of varietal deterioration. The French Acadians, on the other hand, appeared to prefer a naked form over a fuzzy form. In their primitive phase of culture and home manufacture the naked seeds were more easily removed in hand ginning than the fuzzy or tight seeds. Also their variety, along with the recessive conditions of naked seed, carried a higher lint percentage than the inhibitor type of naked seed. Apparently the latter has a strong epistatic effect on normal lint development. The brown lint in the Acadian variety was also of advantage in a primitive culture. The necessity of the use of dyes was avoided. In any crude breeding procedures the Acadians may have practiced, the recessive form doubtless was found fairly easy to keep pure.

Summary

All naked seed × fuzzy seed crosses in Upland cotton heretofore have shown naked to be dominant over all degrees of fuzziness. In a naked seed Upland variety, also having brown lint, found among the French Acadians of Louisiana the naked character is, on the contrary, recessive to fuzzy seed. In F_1's from Acadian Brown separately crossed with eight fuzzy seed varieties the seeds in each case were fuzzy. The fuzzy condition, however, was not completely dominant showing a range of fuzz pattern from that of full and complete fuzziness to that of a pronounced tuft on hilum end and some thin patches of fuzz hair on the raphe and chalazal end.

The previous work in the study of dominant naked and recessive fuzzy has indicated naked as being a simple domi-

nant character and not carrying a suppressed fuzzy gene. On the other hand the 13:3 ratio obtained from crossing dominant naked and recessive naked, and comparing the result with crosses between certain white breeds of poultry, suggests that naked dominance heretofore found in Upland cotton is due to an inhibitor epistatic to fuzz development rather than to a pure condition of a dominant gene itself. The naked character in Acadian Brown in contrast appears to be conditioned by a single recessive gene.

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Hen Into Rooster

IN 1946 the following case of apparently complete sex reversal in fowls was observed on the farm of Walter M. Arnsdorf, located at Glen Mills, Pennsylvania. The importance of the phenomenon was not realized until the case of sex reversal, reported by Crew* was called to the attention of the writer.

Crew had a Buff Orpington which laid eggs three years before any change took place. After molting the fowl developed male plumage, behaved as a male, and proved to tbe fertile when placed with a virgin Buff Orpington hen. The reversal was believed due to abdominal tuberculosis.

During the month of May, 1946, it was noticed that a particular female fowl, that we may call "X," was actually going through the process of mating with other female fowls of the flock. "X" was a New Hampshire red, which previous to this change had what could be called a normal rate of egg production, and during the time that the noticeable change was taking place, "X" was removed from the trap nests several times, but only one egg could be accounted to her. By the month of June "X" did not visit the nests as before, but instead developed many of the male secondary characteristics such as a large, heavy comb, longer spurs, and heavier tail plumage, and even some half-hearted attempts to crow were made.

During the last week of August, it was decided to set some eggs from a hen that "X" had actually been observed mating with, in order to establish their fertility. After twenty-four days two of the original seven eggs placed under a broody hen, hatched out. It must be emphasized at this point that "X" was the only "rooster" that the flock had ever known.

Our practice in trapnesting makes it quite certain that "X" began life as a reasonably reproductive hen. Our egg production records are on a week-to-week basis to determine the number of eggs each hen lays during a week. If a hen falls below a certain standard, she loses her head. "X" must have had an egg production close to the standard or else we would not have kept her as long as we did. This standard varied between three and four eggs a week, depending on the condition of the flock.

Robert E. Arnsdorf
Glenn Mills, Pa.

*Crew, F. A. E. *Proc. Roy. Soc.* 95:256. 1923.

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